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ARE ENVIRONMENTAL CHEMICALS CAUSING MEN TO LOSE THEIR FUNDAMENTAL MASCULINITY?

Are environmental chemicals causing men to lose their masculine characteristics?

A recent study in the BRITISH MEDICAL JOURNAL concludes that men in western countries today have sperm counts less than half as high as their grandfathers had at the same age.[1] In addition, the occurrence of cancer of the testicles has increased 3-fold to 4-fold during the past 40 years; and various birth defects of the male reproductive system have increased 2-fold to 4-fold during the same period, including undescended testicles (a condition called cryptorchidism) and a birth defect called hypospadias in which the male urinary canal is open for a variable distance on the underside of the penis. [2]

An article published last month in THE LANCET, another prestigious British medical journal, asks whether these phenomena can all be traced to the same cause, namely exposure of males very early in life to female sex hormones (estrogens) or to environmental chemicals that act like estrogens.[3]

This hypothesis is being taken seriously within the scientific community; both SCIENCE magazine and C&EN [CHEMICAL & ENGINEERING NEWS] reported on the publication of the LANCET article. [4,5]

The report on sperm counts, in the BRITISH MEDICAL JOURNAL, examined 61 separate studies of sperm count in men in many countries, including the U.S., and concluded that, among men in western countries, there has been a 42 percent decrease in average sperm count, from 113 million per milliliter (ml) to 66 million per ml, since 1940. (There are 4.5 milliliters in a teaspoon.) Furthermore, the average volume of semen diminished from 3.4 ml to 2.75 ml, a 20 percent loss since 1940. Thus the average man

has lost 53 percent of sperm production since 1940.

The researchers examined the possibility that sperm counting methods have changed during the past 50 years, or that racial or geographic factors might be at work. After careful analysis, they concluded that the diminished sperm count in men is real, is widespread throughout the industrialized western world, and affects men of all races.

Some of these facts have been known to some medical researchers for the better part of a decade, but only recently has the explanation been offered that all these problems may be related to male exposures to female hormones (estrogens) early in life.

The hypothesis put forth in THE LANCET last month suggests that males are being exposed in the womb to female sex hormones that permanently alter their sexual development, increase their risk of having undescended testicles, hypospadias, and testicular cancer, and reduce by half the average man's ability to produce sperm.

Five sources of increased estrogenic exposures to males in the womb are being considered:

1) A modern diet may increase the levels of natural estrogen in women. Fiber in the diet today is lower than it was 50 years ago. (Fiber in the diet is basically anything that cannot be digested.) Natural estrogens excreted in the bile are more readily reabsorbed into the bloodstream when the lower intestine contains little dietary fiber. Therefore, a fetus today may be exposed to higher levels of the mother's own natural estrogens, compared to a fetus 50 years ago.

2) Some 3 to 4 million women were treated with a potent synthetic hormone called diethylstilbestrol (commonly known as DES) from 1950 through 1970. Daughters of DES-treated women have an increased risk of a rare vaginal cancer. The sons of DES-exposed women have low sperm counts, and a higher-than-normal risk of malformations of the reproductive tract such as hypospadias and undescended testicles. Furthermore, all these effects can be reproduced in the laboratory by exposing mice and rats to DES. Thus there is compelling evidence, from humans and other animals, that males exposed in the womb to female hormones can suffer reproductive system damage, some of which only becomes apparent after puberty in the form of reduced sperm count.

3) Synthetic estrogens, including DES, were fed to beef cattle from the 1950s through the 1970s to make them grow more meat faster. Such practices may have increased the quantity of estrogens in meat-eating women and perhaps, as a contaminant, in some water supplies.

4) The use of synthetic estrogens as a contraceptive pill has increased greatly during the past 20 to 40 years. One such compound, ethinyl estradiol, has been detected as a contaminant in some water supplies, but the data are skimpy.

5) Another source of increased estrogens in women today is the many synthetic organic chemicals and

heavy metals that have been released into the environment in massive quantities since world war II. Some of these compounds, such as PCBs and dioxins, are known to interfere powerfully in the reproductive system of fish, birds, and mammals, including humans.[6] A single, tiny oral dose [0.064 micrograms per kilogram of body weight] of dioxin on day 15 of pregnancy in rats has no effect on the mother but increases the likelihood of various reproductive disorders in their male offspring: undescended testicles, smaller testicles, reduced levels of male hormone circulating in their blood, and reduced sperm count.[7] Here again, we see effects caused by exposures in the womb, but which only become apparent after the offspring mature. Many common industrial chemicals are weakly estrogenic, [8] but they are now present in all our food and water, and are stored in the fat tissues of our bodies, including women's breast milk. As one researcher observed, "Humans now live in an environment that can be viewed as a virtual sea of oestrogens."[9] [Estrogens and oestrogens are the same thing, only spelled differently.]

These findings and hypotheses add to the growing body of medical knowledge indicating that many chemicals --especially chlorinated hydrocarbons --mimic hormones and interfere with the endocrine systems of fish, birds, wildlife and humans. Earlier studies have linked chlorinated hydrocarbons to female breast cancer [see RHWN #279, #334], and it is worth pointing out that breast cancer in women is associated with an increased likelihood of testicular cancer in their sons. [10] Another well-established risk factor for testicular cancer is undescended testicles. [11] Thus breast cancer, testicular cancer, and defects of the male reproductive system, including diminished sperm count, all seem linked.

Diminished sperm count alone is a potentially serious matter. Many animals produce up to 1400 times as much sperm as is needed for fertility. In contrast, the average human male produces only 2 to 4 times as much sperm as is needed for fertility. [12] Humans don't have much sperm to spare. A 50 percent reduction in human sperm count may thus diminish human fertility and could therefore take away from men the one thing they indisputably do well: help women propagate the species.
--Peter Montague, Ph.D.

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[1] Elisabeth Carlsen and others, "Evidence for decreasing quality of semen during past 50 years," BRITISH MEDICAL JOURNAL Vol. 305 (1992), pgs. 609-613.

[2] A. Giwercman and N.E. Skakkebaek, "The human testis--an organ at risk?" INTERNATIONAL JOURNAL OF ANDROLOGY Vol. 15 (1992), pgs. 373-175. And: A. Osterlind, "Diverging trends in incidence and mortality of testicular cancer in Denmark, 1943-1982," BRITISH JOURNAL OF CANCER Vol. 53 (1986), pgs. 501-505.

[3] Richard M. Sharpe and Niels E. Skakkebaek, "Are oestrogens involved in falling sperm counts and disorders of the male reproductive tract?" THE LANCET Vol. 341 (May 29, 1993), pgs. 1392-1395. And see: R. M. Sharpe, "Declining sperm counts in men --is there an endocrine cause?" JOURNAL OF ENDOCRINOLOGY, Vol. 136 (1993), pgs. 357-360.

[4] Constance Holdren, "The Hazards of Estrogens," SCIENCE Vol. 260 (May 28, 1993), pgs. 1238-1239.

[5] "Estrogenic Chemicals May Lower Sperm Counts," C&EN [CHEMICAL & ENGINEERING NEWS] June 7, 1993, pg. 28.

[6] Glen A. Fox, "Epidemiological and Pathobiological Evidence of Contaminant-Induced Alternations in Sexual Development of Free-Living Wildlife," in Theo Colborn and Coralie Clement, CHEMICALLY-INDUCED ALTERATIONS IN SEXUAL AND FUNCTIONAL DEVELOPMENT: THE WILDLIFE/HUMAN CONNECTION (Princeton, N.J.: Princeton Scientific Publishing Co., 1992), pgs. 147-158. And: Peter J.H. Reijnders and Sophie M.J.M. Brasseur, "Xenobiotic Induced Hormonal and Associated Developmental Disorders in Marine Organisms and Related Effects in Humans," in Theo Colborn and Coralie Clement, CHEMICALLY-INDUCED ALTERATIONS IN SEXUAL AND FUNCTIONAL DEVELOPMENT: THE WILDLIFE/HUMAN CONNECTION (Princeton, N.J.: Princeton Scientific Publishing Co., 1992), pgs. 159-174.

[7] Thomas A. Mably and others, "IN UTERO and Lactational Exposure of Male Rats to 2,3,7,8-Tetrachlorodibenzo-P-dioxin. 3. Effects on Spermatogenesis and Reproductive Capability." TOXICOLOGY AND APPLIED PHARMACOLOGY Vol. 114 (May, 1992), pgs. 118-126.

[8] Theo Colborn and Coralie Clement, CHEMICALLY-INDUCED ALTERATIONS IN SEXUAL AND FUNCTIONAL DEVELOPMENT: THE WILDLIFE/HUMAN CONNECTION (Princeton, N.J.: Princeton Scientific Publishing Co., 1992), pgs. 1-2, list the following chemicals "known to disrupt the endocrine system:" DDT and its degradation products [DDE and DDD], DEHP (di(2-ethylhexyl) phthalate), dicofol, HCB (hexachlorobenzene), kelthane, kepone, lindane and other hexachlorocyclohexane congeners [forms], methoxychlor, octachlorostyrene, synthetic pyrethroids, triazine herbicides, EBDC fungicides, certain PCB congeners [forms], 2,3,7,8-TCDD and other dioxins, 2,3,7,8-TCDF and other furans, cadmium, lead, mercury, tributyltin and other organo-tin compounds, alkyl phenols (non-biodegradable detergents and anti-oxidants present in modified polystyrene and PVCs), styrene dimers and trimers, soy products, and laboratory animal and pet food products."

[9] Richard M. Sharpe and Niels E. Skakkebaek, cited above in footnote 3, quoting B. Field and others, "Reproductive Effects of Environmental Agents," SEMINARS IN REPRODUCTIVE ENDOCRINOLOGY, Vol. 8 (1990), pgs. 44-54.

[10] A. R. Moss and others, "Hormonal risk factors in testicular cancer; a case control study," AMERICAN JOURNAL OF EPIDEMIOLOGY Vol. 124 (1986), pgs. 39-52.

[11] M.B. Jackson and others, "The Epidemiology of Cryptorchidism," HORMONE RESEARCH Vol. 30 (1988), pgs. 153-156.

[12] Peter K. Working, "Male Reproductive Toxicology: Comparison of the Human to Animal Models,"

ENVIRONMENTAL HEALTH PERSPECTIVES Vol. 77 (1988), pgs. 37-44.

Descriptor terms: sperm count; fertility; cancer; birth defects; statistics; undescended testicles; cryptorchidism; hypospadias; estrogens; food safety; des; diethylstilbestrol; cattle; agriculture; beef; contraceptives; birth control; the pill; ethinyl estradiol; metals; chlorinated hydrocarbons; chlorine; breast cancer;

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